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SOME PROBLEMS IN THE STUDY OF HEREDITY IN MENTAL DISEASES

By

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SOME PROBLEMS IN THE STUDY OF HEREDITY IN MENTAL DISEASES.*

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Without doubt, from the standpoint of prophylaxis, at least, the most important aspect of psychiatry has always been the factor of heredity. It has been known for years, in a general way, that heredity has played an important rôle in the etiology of the psychoses, but we have been very far from having any definite knowledge of the subject. We have, until quite recently, been satisfied with vague opinions regarding "insanity in the family," of those mentally affected, without due regard to the nature of the malady in other members of the family.

The records of the patients in insane hospitals, even in those hospitals pretending to do modern scientific work, are woefully incomplete, inadequate and often inaccurate. In the older methods of examination the statistical data regarding heredity, as well as etiology in general, was based upon the statements of the committing physician. No attempt was made to inquire systematically into these questions, and every one knows how unreliable such statements received must have been. Even with modern methods, but very little progress was shown, as the source of information from the families of patients was also limited. The most conscientious work of the assistant physicians would fail to bring out all the important factors of heredity. Often the husband or wife of a patient was the only one who came to visit the patient, and usually they knew very little of the family history of each other. Hence, statistics made up from such sources, while much more accurate and complete than statistics collected as above, at the same time the best obtainable by this method were open to serious and just criticism.

* Elaboration of a paper read by title at the sixty-seventh annual meeting of the American Medico-Psychological Association, Denver, Colo., June 19-22, 1911.

We have been content until quite recently with the loose methods of investigating this important subject, and the fact that insanity occurred in the family in such and such a proportion of our patients was considered enough for our present knowledge.

It is usual to see in statistics of insane hospitals a summary of the number of cases in which heredity was a factor, such heredity being merely insanity in the family. We have been aware that certain forms of insanity exhibited more heredity than others. But we have had no accurate knowledge of the nature and type of mental disease in ancestors and immediate families of our patients. We must all plead guilty to our lack of interest in this subject, and we must acknowledge our indebtedness to one outside of our work who has succeeded in arousing our interest and stimulating our endeavors in this field.

Prof. Charles B. Davenport, of the Eugenics Section of the American Breeders' Association, and in charge of the Eugenics Record Office at Cold Spring Harbor, has been the one to stimulate our interest in this field.

In this office, in which Prof. H. H. Laughlin has been associated with Prof. Davenport, the modern ideas of the study of heredity had its birth. The office has been in existence less than three years, but has already accomplished a great deal, and through this agency many state hospitals, as well as other institutions for the care of epileptics and the feebleminded, have been supplied with competent and well-trained field workers, who are now engaged in vigorously attacking the problem in a score of centers. The workers have been prepared by means of a summer school, which lasts six weeks. During this time systematic instruction is given in collecting data in the field, tabulating such data, and in making heredity charts. The workers are usually chosen from among college graduates, and those having some experience in social service work, preferably women.

It is with some pride that we note that New Jersey has been the pioneer in this special line of work. The first systematic study of the question of heredity in the feebleminded was the work of Prof. Johnstone and Dr. Goddard, of the Training School, at Vineland, and Dr. Weeks, of the Epileptic Village, at Skillman, has contributed the first important contribution based upon systematic field work on the question of heredity in this class of patients.

Dr. Everett Flood, of the Epileptic Colony, at Monson, Mass., has also done valuable work in this field. These can be said to have been pioneers in this work.

The King's Park State Hospital, in the State of New York, was the first state hospital for the insane to employ field workers in connection with their study of heredity, but such work has not been continued. We believe the New Jersey State Hospital at Trenton was the first to organize a permanent department of "field work," with a special appropriation to carry on the work, in connection with systematic "after care" work, and has now two trained field workers. By combining the heredity work with the "after-care" work, we feel that a direct benefit comes to the patient, and that we can show results of a practical immediate character as well as results which might be unjustly termed of theoretical importance only.

Literature.

Although Gregor Mendel published his discoveries of laws regarding heredity, which now bear his name, and are so well known, as early as 1866, it was not until 1900, when his work was rediscovered by De Vries and others, that the importance of his work in this field was recognized. It was mainly through the work of Bateson and other Englishmen that his work received the recognition in England and this country. Since 1900 many investigators have been occupied with the problems of heredity, especially in plant and animal life. To some extent the problems of human heredity have been investigated, and the science of eugenics has now grown to considerable proportions and found an important place in our studies of the human family.

But the relation between the science of eugenics and psychiatry has but recently been established, and the literature upon the subject is as yet extremely meagre, and, with one or two exceptions, is confined exclusively to this country. In only one other country can it be said that systematic study of heredity in insanity by means of field work has been established, and that is Germany. It is all the more surprising that this country, where so much valuable research in psychiatry had been carried on, had neglected this phase of the subject, especially as Mendel was a German, and his work should have been familiar.

And up to the present only one investigator has been occupied in this field in Germany, Dr. E. Rüdin, Oberartz of the Royal Psychiatric Klinic in Munich, under the direction of Prof. Kraepelin. His work is decidedly a most important contribution to the subject. His work is of such importance to the study of heredity in mental diseases that it is worth our while to review it more in detail, which will be done later. Since 1909, Rüdin has been Oberartz of the Psychiatric Klinic at Munich, and during this period he has personally collected material for the study of the complex problems of this question. (This valuable contribution of his is published in the Zeitschrift fur Gesammte Neurologie and Psychiatrie, Seventh Vol., 5th Part, Nov. 18, 1911.)

What makes the work of Rüdin so remarkable and noteworthy is the fact that he has done field work personally, and at his own expense. He gives up his position at the clinic for several months each year and goes into the field to collect this data. He has copies of the rosters of all the Bavarian institutions, and is able to get fairly good records of the patients who were in these institutions, although patients were committed a great many years ago. The fact that he has access to accurate records is a great advantage, especially in case of preceding generations.

From the majority of records of the hospitals in this country, even up to within a short time ago, it would be impossible to make any sort of a diagnosis of the patients admitted to them. In some cases one can get more accurate information upon which to base a diagnosis from the description of the cases who never come into the hospital than in patients who are admitted.

Another fact of importance connected with Rüdin's work is that he does his field work himself, and, consequently, is able to get better descriptions, to observe, and to " size up " the members of the family and classify them accordingly. Without financial assistance, and against difficulties which would seem insurmountable to us, he has achieved some wonderful results. He has accurate family histories in a great number of cases, representing various types of mental disease. Although he has so much material at his disposal, he feels that he has not done enough work to justify any general statements or formulation of laws regarding the heredity factors of insanity.

The Eugenics Record Office has issued bulletins from time to time, based upon the work done by their field workers in various institutions.

The first bulletin is on "The Heredity of Feeblemindedness," by H. H. Goddard, Ph. D., of the Training School, at Vineland, and is an extremely interesting and valuable contribution to the subject. Fifteen charts are shown. While it is a preliminary report of the work being done at Vineland, it is well worth reading by those interested.

Bulletin No. II, from the same office, is a compilation by Dr. Davenport, Prof. H. H. Laughlin, Dr. Weeks, Prof. Johnstone and Dr. Goddard, on the "Study of Human Heredity," and methods of collecting, charting and analyzing data. It has been the object of those interested in this subject to have a uniform method of charting which could be adopted by all institutions, so that the various institutions would understand, without difficulty, the work that is being done in other institutions.

Bulletin III is by Gertrude L. Cannon and A. J. Rosanoff, M. D., of the King's Park State Hospital, and is a preliminary report of the work done at the King's Park State Hospital, New York. This work is principally an outline of the methods used, and a brief description of the Mendelian laws.

The fourth bulletin, by Drs. Weeks and Davenport, is the result of systematic field work at the Epileptic Village, and is a valuable contribution to the inheritance of epilepsy. The authors give the following conclusions as the result of their work:

1. The method of field-study of epileptic families combined with the modern biological methods of analysis of hereditary data constitute a vastly improved means of inquiry into inheritance of epilepsy.

2. Epilepsy and feeblemindedness show a great similarity of behavior in heredity, supporting the hypothesis that such is due to the absence of a protoplasmic factor, that determines complete nervous development.

3. When both parents are either epileptic or feebleminded, all their offspring are so likewise.

4. The conditions, named migraine, chorea, paralysis, and extreme nervousness, behave as though due to a simplex condition of the protoplastic factor that conditions complete nervous development; *i. e.*, persons belonging to these classes usually carry some wholly defective germ cells. Such persons may be called "tainted."

5. When such a tainted individual is mated to a defective, about one-half of the offspring are defective.

6. When a simplex normal is mated with a defective, about half the offspring are normal; the others defective or neurotic.

7. When both parents are simplex in nervous development and "tainted," about one-quarter (actually 30 per cent) are defective.

8. The proportion of tainted offspring is not noticeably higher when both parents show the same nervous defect.

9. Normal parents that have epileptic offspring usually show gross nervous defect in their close relatives.

10. While we recognize that "epilepsy" is a complex, yet there is a classical type numerically so preponderant that, in the mass, "epilepsy" acts like a unit defect.

11. Our data point to a poisoning in slight degree of germ cells by alcohol, but the evidence is hardly crucial.

12. There is evidence that in epileptic strains the proportions of epileptic children in the latest complete generation is double that of the preceding; but there is no evidence that in these epileptic strains the average number of children in a fraternity is greater than in the population at large. Provided matings continue as at present, and no additional restraint is imposed, the proportion of epileptics in New Jersey would double every thirty years.

13. The most effective mode of preventing the increase of epileptics that society would probably countenance is the segregation during the reproductive period of all epileptics.

Dr. Rosanoff and Florence I. Orr, B. S., are the authors of Bulletin No. V, entitled, "The Study of Heredity in Insanity in the Light of the Mendelian Theory." The conclusions of the authors are based upon the investigation of about 73 cases, and the heredity charts in these cases are reproduced. This represents 206 different matings, total, 1097 offspring. A table is given, showing the proportion of normal and neuropathic offspring, which resulted from various types of matings, compared to the theoretical expectations according to the Mendelian theory. The authors have considered that the neuropathic constitution, in reality, consists of a series of units, which are distinct, at least from the standpoint of clinical definition, though at the same time in manner related to each other. One is forced to emphasize here the fact that it is necessary to keep an open mind in regard to these problems. In other words, that we must investigate the facts of the heredity of psychoses as they exist, and not to be too

prejudiced towards the Mendelian laws. Following are the conclusions given by the above authors:

1. The neuropathic constitution is transmitted from generation to generation in the manner of a trait, which is, in the Mendelian sense, recessive to the normal condition. Rules of theoretical expectation are accordingly as follows:

a. Both parents being neuropathic, all children will be neuropathic.

b. One parent being normal, but with the neuropathic taint from one grandparent, and the other parent being neuropathic, half the children will be neuropathic and half will be normal, but capable of transmitting the neuropathic make-up to the progeny.

c. One parent being normal and of pure normal ancestry and the other parent being neuropathic, all the children will be normal, but capable of transmitting the neuropathic make-up to their progeny.

d. Both parents being normal, but each with the neuropathic taint from one grandparent, one-fourth of the children will be normal and not capable of transmitting the neuropathic make-up to their progeny, one-half will be normal, but capable of transmitting the neuropathic make-up, and the remaining one-fourth will be neuropathic.

e. Both parents being normal, one of pure normal ancestry and the other with the neuropathic taint from one grandparent, all the children will be normal, half of them will be capable, and half not capable of transmitting the neuropathic make-up to their progeny.

f. Both parents being normal and of pure normal ancestry, all the children will be normal and not capable of transmitting the neuropathic make-up to their progeny.

2. Various clinical neuropathic manifestations bear to one another the relationship of traits of various degrees of recessiveness; in a most marked way recoverable psychoses, though recessive as compared with the normal condition, are dominant over epilepsy and allied disorders.

3. Various other clinical neuropathic manifestations bear to one another the relationship of neuropathic equivalents; that is to say, they are conditions of the same degree of recessiveness, varying in their clinical manifestations with the personality of the subject, environmental conditions, etc.

4. All the neuropathic children, which result from a mating of the fourth type (both parents normal, but each with the neuropathic taint from one grandparent), can have, theoretically, only equivalent defects and not defects of different degrees of recessiveness.

5. Among the actual results from such matings the following have been met with:

a. Brothers and sisters suffering from clinically identical neuropathic manifestations.

b. Psychosis in one subject and peculiar or abnormal disposition, but no actual psychosis in brothers and sisters.

c. Psychosis in one subject and isolated, but clinically related symptoms in brothers or sisters; we find with particular frequency dementia præcox—fainting spells or convulsions in childhood.

d. Psychoses clinically not known to be related; senile deterioration—peculiar hysteriform psychoses.

6. Neuropathic conditions show only in about one-fourth of the cases indications for commitment to sanitariums or public institutions. The total incidence or neuropathic conditions may be roughly estimated as affecting between 1.5 and 2 per cent of the general population.

7. It is further estimated that about 30 per cent of the general population, without being actually neuropathic, carry the neuropathic taint from their ancestors and are capable under certain conditions of transmitting the neuropathic make-up to their progeny.

The methods of assuming facts, when they do not exist, is open to just criticism, and the fact that it was necessary for the authors to assume the fact of the simplex inheritance in places where information was not available, is open to criticism. It is true that these cases where the simplex inheritance is dissimilar has been treated separately and distinct from the other material. It is self-evident that the question of human inheritance presents many difficult problems, and there will be many cases that apparently do not follow any given law. The lack of matings and the absence of children in many families, or the usual " two-children " families, offers serious difficulties in making out definite laws regarding inheritance.

To some extent feeblemindedness can be considered a unit, but here one is forced to recognize the fact that imbecility itself is far from being a unit, and may be caused by entirely different factors. The grades of feeblemindedness, as outlined by Goddard, viz.: highest types, morons, next grade, feeblemindedness, and then imbecility and idiocy, are practical for a clinical classification, but from the standpoint of etiology these types may not be so distinct, and there certainly can be a distinct difference between the members of any one class. No one will deny that in feeblemindedness we have the purest form of inheritance, and that feebleminded parents, as found by Dr. Goddard, will certainly produce feebleminded children. At the same time, a no small number of cases of feeblemindedness may be the result of external causes, and not altogether due to heredity features. Infectious diseases in child-

hood, especially scarlet fever, and perhaps other fevers, cause feeblemindedness, both directly and indirectly, by arresting development through the direct action upon the cerebral tissues, and indirectly produce arrested development through deafness and other disturbance of the sensory organs.

When discussing the cause of epilepsy, one has to be especially careful not to consider this disease as a unit, a fact recognized by Weeks and Davenport. It is far better to consider the group as "the epilepsies" rather than to consider the disease as a unit. So it is even necessary, in discussing the inheritance of these simple forms of disease, to be guarded in not considering them entirely as units. At the same time, we recognize the fact that inheritance plays an important part in the production of feeblemindedness and "the epilepsies" and that the laws concerning same will be much simpler than the laws regarding other psychoses. In fact, we are forced to consider that these two diseases are subdivisions of insanity as such, and for this reason one readily sees the error in considering insanity as a unit or uniform disease.

F. W. Mott (Brain, Part 2-3, Vol. XXXIV, Nov., 1911), "Inborn Factors of Nervous and Mental Diseases," discusses the question of inheritance in general, and in particular the hereditary features of insanity. He discusses at length the laws of Galton, and is inclined to agree with his views, which laws, as we know, are opposed to those of Mendel. He also discusses the Mendelian principals at length, and gives a detailed explanation of these laws. He firmly believes in the law of sex limitation in certain types of diseases, such as color blindness and hæmophilia, and, in the field of nervous diseases, pseudohypertrophic paralysis. This form of inheritance is not only discontinued or interrupted for successive generations, but the disease is limited to one sex, although it is to be noted that the disease is transmitted by the sex in which it does not appear. Thus, it is the males who are affected in hereditary sex-limited diseases, and it is the females who transmit the disease. Mott also gives considerable space to "Nature and Nurture," and shows, conclusively, how in a great many conditions the environment and experience due to environment may have a very important bearing on inheritance. Also that a neurotic tem-

perament may be manifested in many different ways by conduct and behavior, and this neurotic temperament may be the first evidence of any degeneration in the stock. It is well that he has emphasized this important fact, for these characteristics must be looked for in collecting data for pedigrees of the insane, as it has been found that they are of as much importance as the pure mental disease in the ancestors. It is true, as he states, " that unsound stock may have successful men in the eyes of the world, but these may really form the first step in the process of degeneration, for avarice and normal guile, which made them pillars of society, may come out in the next generation as gross criminality or insanity. Mott is of the opinion that inborn factors partly, if not wholly, can account for the appearance of insanity in the stock."

Of considerable interest is the discussion of the Law of Anticipation, which was defined by Nettleship as " a manifestation of the morbid change at an earlier period of life, either in members of each succeeding generation as a whole, or as successively born children of one parentage." He gives examples of the truth of such a law. His observation, " that there is a general tendency for insanity not to proceed beyond three generations, either because of regression to the normal, or from the fact that the stock dies out," is important. But his explanation, that not infrequently the stock dies out through the inborn tendency of insanity manifesting itself in the form of congenital types, such as imbecility, or in the insanity of adolescence, is open to criticism, for it is not always true that children of insane parents are defectives. Types of insanity, of course, have to be considered, but even children of dementia præcox are frequently entirely normal, and the brothers and sisters of such patients may also be normal, although many of them appear to be peculiar. Mott gives some interesting statistical data regarding familial character of insanity, but here one is forced to call attention to the uselessness of such statistics, where insanity is considered as a unit. We call attention elsewhere to the necessity of considering various types separately, at least until we can establish some sort of relation between the various forms, especially as regards hereditary features. He gives statistics of 2246 individuals where one or more members of the family were inmates of an institution. This is all right as far as it goes, but

in our field work we frequently find evidences of mental disease in members of the family where these individuals have never been inmates of an institution. Especially is this true in early generations, where a very small percentage of those who were insane have been committed to a hospital. So that, to be accurate, one must consider these cases as well as members of the family who have been in institutions. It is also important to note that statistics based on hospital admissions alone would not truthfully represent the facts.

Mott gives the conclusions of Dr. Edward Shuster, who made the study of inheritance of the same types of insanity in 1910. They are as follows:

1. A periodically insane son or daughter is more likely to be associated with a periodically insane mother or father than if one if differently affected. In the case of two offspring in the insane there is even a greater tendency for a periodically insane male or female to be associated with a periodically insane brother or sister than with one differently affected.

2. In case of delusional insanity, the tendency for the affection to run in families is very strongly marked, and the correlation between members of the same co-fraternity is more strongly marked than between parents and offspring.

3. In the instance of primary dementia of adolescence, there is a strong correlation between members of the same co-fraternity. There is also a decided tendency indicated for the brothers and sisters of imbeciles to be also imbeciles.

4. There is no indication of general paralysis running in families. This is not surprising, as it is now recognized to be an acquired disease due to syphilitic infection. Both conclusions would seem to be justified from our knowledge at the present time. The chief criticism of Mott's work is that he has not gone carefully enough into the question, as he has practically taken only the cases which have been admitted to the institutions as a basis for his statistics, thus leaving out of consideration a large number of important individuals.

It is conclusively demonstrated in this country that only by the help of well-trained field workers can we expect to collect valuable data regarding this complex question, and, secondly, in considering the inheritance of insanity, he is inclined to treat the disease as a unit, rather than to closely differentiate the various types. We must first establish the same rules between the various types of psychoses before we can justly consider them similar or dissimilar as regards the form of inheritance. Mott's article, in all prob-

ability, represents the best work that has been done by the English in this field.

Mendelian Laws in Relation to Insanity.

In recent literature we find explanations of the principles of heredity as formulated by Mendel, but the clearest exposition of the subject is found in the work of Rüdin.

We are acquainted with the facts that the total inheritance of an individual from his parents is certain human characteristics, each of which is inherited independently of all the rest, and the inheritance of any such character is believed to be dependent on the presence in the germ plasm of a substance called the determiner.

With reference to any given character the condition of an individual may be dominant or recessive: the character is dominant when, depending upon the presence of its determiner in the germ plasm, it is plainly manifest. It is recessive when, owing to the lack of its determiner in the germ plasm, it is not present in the individual under consideration.

The symbols *D* and *R* in the following table represent the dominant and recessive conditions. In other words, *D* stands for the presence of the determiner of the trait, and *R* stands for its absence.

The following formula for six types of matings and their resulting offspring:

Type 1. $(D+D)X(D+D)=4DD.$
Type 2. $(D+D)X(D+R)=2DD+2DR.$
Type 3. $(D+D)X(R+R)=4DR.$
Type 4. $(D+R)X(D+R)=DD+2DR+RR.$
Type 5. $(D+R)X(R+R)=2DR+2RR.$
Type 6. $(R+R)X(R+R)=4RR.$

We speak of the inheritance of a character from both parents as duplex inheritance, designated by *DD*.

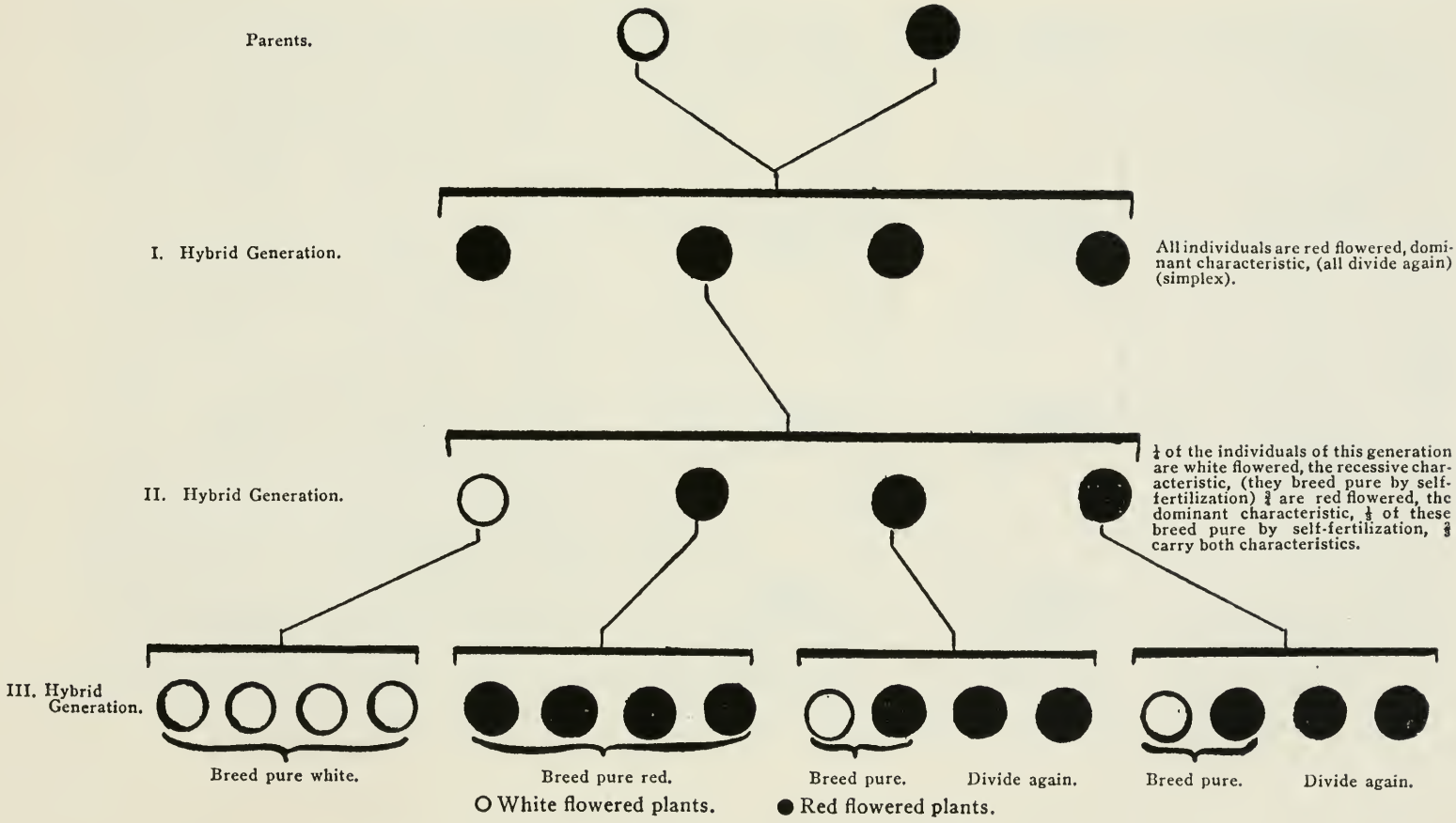
The case of inheritance of a character from one parent is spoken of as simplex inheritance, designated by the symbol *DR*.

In Fig. I, taken from Rüdin, we have exhibited diagrammatically the principles of the Mendelian prevalence and the rules of

FIG. I.

MENDELIAN PREVALENCE AND RULES OF DIVISION.

(Inheritance of the pea flower.)

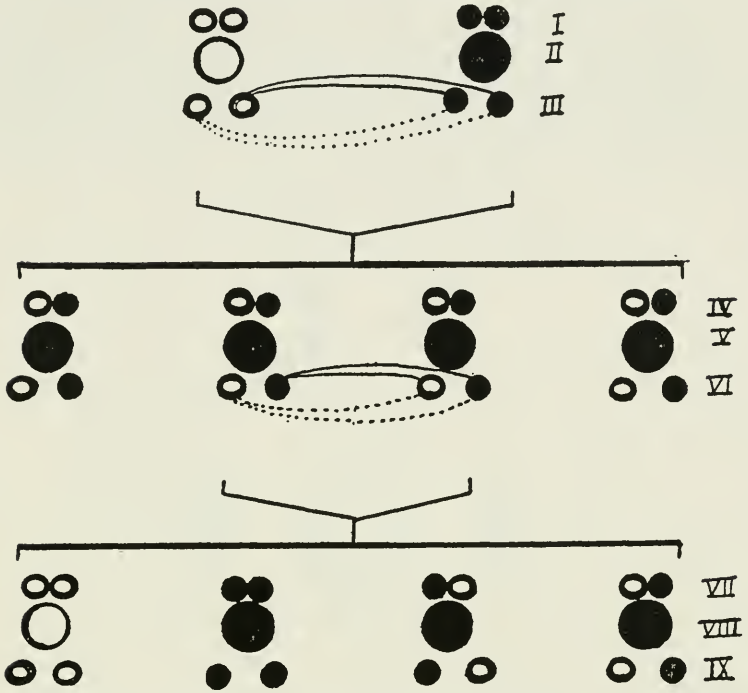


variance in the original experiment of the crossing of red and white peas. In the first hybrid generation we see that all individuals are red-flowered peas, although this generation contains also white-flowered characteristics, which are not manifest because the red is dominant. In the second hybrid generation we see the Mendelian proportions of one white to three red, in other words, one recessive and three dominant. In the third hybrid generation the white sweet pea breeds only white, one-third of the red peas breed pure red, while two-thirds of these red peas breed white and red in the proportion of one to three. This is the simplest explanation of this law. We see by this chart that the pure Mendel inheritance is not a mixed product composed of inherited characteristics, and these characteristics do not exist in a permanent combination, but always occur as separate unchanged characters in succeeding generations.

In Fig. II (after Rüdin), we see a diagrammatic explanation of this law of the regular appearance of the dominant and recessive characteristics. We see in Row I the germ plasm from which the parents develop. On the right the two black dots represent the homozygous gameten, because the germ plasm or the fertilized germ cells, gameten, has only one characteristic, that is, either for white or red. The germ plasm of these separate individuals, *i. e.*, the father and mother, are also considered as pure homozygous. By mating this pair, and following the lines in Row III, we will see there is an equal number of red and white gameten. In Row IV, which produces offspring shown as red in Row V, but, although this generation is red entirely, the gameten are not homozygous but heterozygous, that is, made up of both red and white characteristics, but because the red is dominant, the white characteristic does not appear. Now, by mating two of this generation, and noting the lines indicating combinations in Row VI, we see the reason for the proportions in the second hybrid generation. We have one white individual made up of homozygous gameten, one red homozygous and two heterozygous. Because of the red being dominant, the white characters do not show. Then in the germ plasm of these individuals we have the white producing pure white, and the red producing pure red, and the heterozygots producing both white and red.

FIG. II. (After Rüdin.)

The Experimental Crossing of the Red and White Flowered Pea.
(The "anlage" combination of the Gametes and zygots.)



○○ = Germ plasm which carries the tendency (anlage) for white flowers only.

●● = Germ plasm which carries the tendency (anlage) for red flowers only.

○● = Germ plasm which carries tendency for red as well as white flowers.

Row I. Germ plasm from which the parents are derived, they carry the one character. White or red, they are pure homozygous.

Row II. The two parents which result from the above germ plasms.

Row III. Pure, homozygous germ plasm which is produced by the parents.

Row IV. Result of the union of the unit characters from the parental germ plasm, heterozygous are combined germ plasms (simplex).

Row V. Here the tendency for red in the germ is dominant over the tendency for white. The flowers appear red, the first hybrid generation.

Row VI. The two characters, which are present in the heterozygous germ plasm and are derived from the parents, divide again, so that half of the germ cells bear the tendency (anlage) for white, the other half the tendency for red.

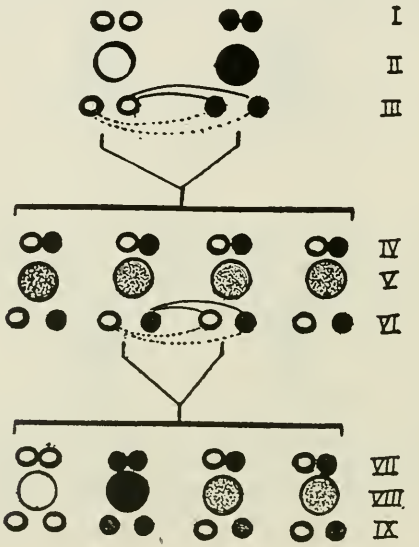
Row VII. The fourth combination of characters which occurs through the crossing of two hybrids of the first hybrid generation, two heterozygous and two homozygous.

Row VIII. Where the tendency for red is dominant over white, red flowered and white flowered individuals result from the four named germ plasms in the proportion of one to three.

Row IX. The above-named combination of germ plasms from the first hybrid generation will again divide. The one white individual, because it is produced from germ plasm with a tendency (anlage) for white only, produces germ cells with a tendency for white; one red individual with germ plasm with tendency for red only and two red individuals with both characters.

There is still another form of inheritance, which is shown in Fig. III, which is known as incomplete inheritance of the dominant characters or intermediate inheritance. This figure illustrates the mating of the red and white " wunder blume " or maribilis jalappa. By mating the red and white plant of this species we get in the

FIG. III. (After Rüdin.)
Scheme of Mendelian Inheritance of the Intermediate Type.



oo Germ plasm, which carries the tendency (anlage) for white flowers only.
●● Germ plasm, which carries the tendency (anlage) for red flowers only.
o● Germ plasm, which carries the tendency for red flowers as well as for white flowers.
O White flowered individual.
● Red flowered individual.
◉ Pink flowered individual.

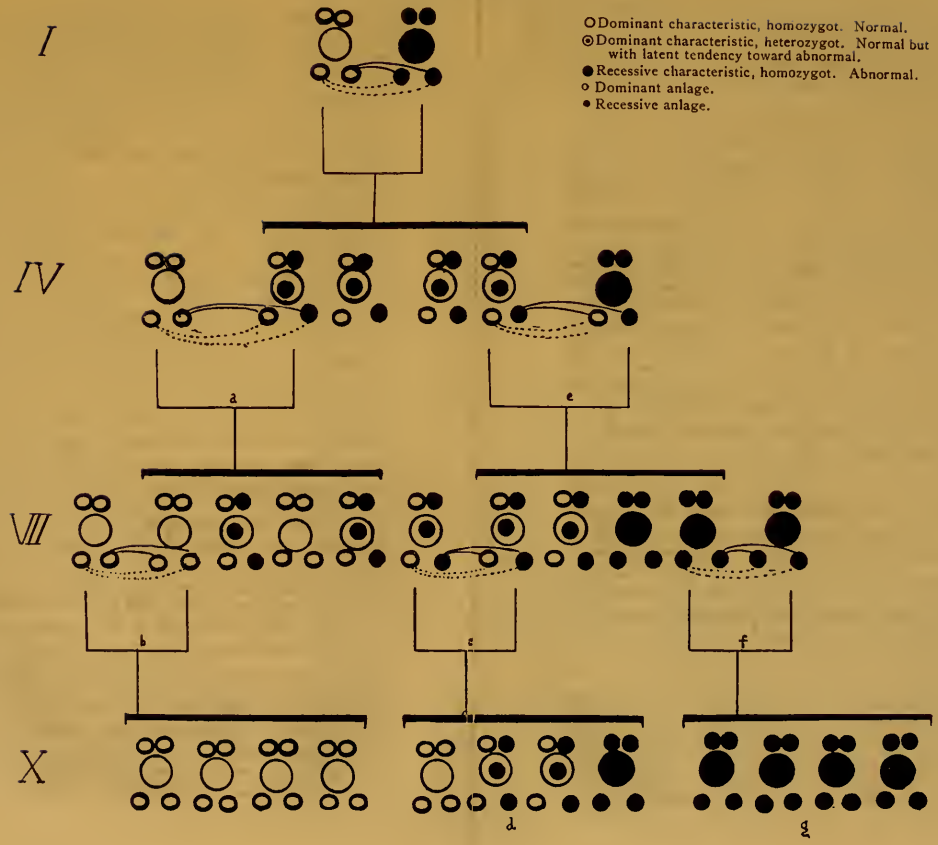
first not pure red or pure white or the prevalence of dominant characters, but the progeny shows a resulting mixture of red and white indicated by pink, but, as shown in Row IV, this progeny-colored pink is made up of heterozygous gameten, capable of producing both red and white. This is shown in the succeeding third generation, where we have one pure white individual, one pure red individual and two pink individuals. The white and red

are homozygous, while the two pink plants are heterozygous, and this explains the fact that while the law of prevalence is an important factor of Mendelian heredity, at the same time the most important fact is that the antagonistic characteristic factors do not produce any permanent combination, but always occur as a separate unchanged character in succeeding generations.

So far we have spoken of the inheritance of dominant characteristics, and this rule holds good when this dominant characteristic is an abnormality.

We are now to consider the laws of inheritance, where the abnormality is recessive. This is shown in Fig. IV. The recessive characteristics here are indicated by the black, hence are not to be confused with the other figures where the black indicates the dominant characters. Here there are six possibilities of a simple recessive Mendelian inheritance. In Row IV we see the result of the mating of a dominant homozygot with recessive heterozygot (Row I). All the individuals in Row IV are normal, notwithstanding the fact that they inherited a defect from one parent. This is illustrated by the white circle with the black dot. The abnormality is not apparent when, for example, the dominant normal mates with the dominant normal (in Row IV-a the dominant homozygot mates with a dominant heterozygot; Row VII-b, the dominant heterozygot mates with a dominant homozygot). There is a very important exception when the abnormality again comes to the surface, as seen in Row VII-c, where two dominant heterozygots mates with homozygots, in other words, two normal individuals with a duplex inheritance (normal and abnormal). Then we have one quarter of the progeny recessive homozygot, therefore abnormal in Row X-d. However, when recessive homozygot (abnormals) mates with dominant heterozygot (Row IV-e), then we have one-half of the progeny abnormal, and, finally, when two recessive abnormals, homozygots mate (Row VII-f) all the progeny will be abnormal (Row 10-g). When one of the parents is sick (abnormal), mated to a normal or normal mated to normal individuals, the progeny is normal children. They may also have abnormal children. Normal individuals from affected families will have normal progeny, the same as normal individuals from families without any inherited defect. We have these two phenomena, matings between normal individuals from

FIG. IV. (After Rüdin.)
"Anlagen" Combination of Recessive Abnormalities.



families without hereditary taint will show all normal progeny. Where both parents are abnormal all the progeny will be abnormal.

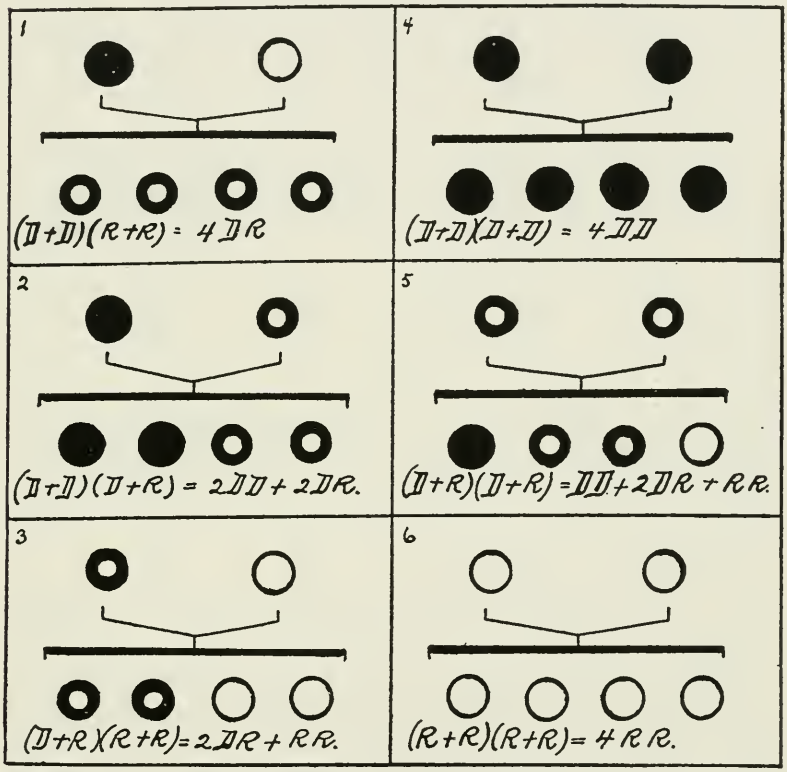
If we analyze the proportions in this chart, we will see that the apparent irregularity, with so-called exceptions, have a definite basis and follow definite rules. When two normal individuals have abnormal children, the proportion is one to four or two to eight, while parents who are normal and well, at the same time their germ plasm is not pure. Instead, the germ plasm is heterozygous. One or the other of the parents comes from ancestors, one of whom at a certain time was abnormal. When normal individuals have normal children, at least one parent has pure germ plasm, *i. e.,* homozygous, whose ancestors have never had any similar disease, or only through indirect ancestors, the grandparents or great-grandparents, or the collaterals, but never from the parents. In cases where an abnormal has normal progeny, the other parent must necessarily be homozygot (normal), and there is no tendency to abnormal. When an abnormal individual mates with a normal individual and have abnormal children, we find that the proportions of the abnormal to the normal will be as one to one, in other words, half of the children will be abnormal. In this case, however, the normal parent is heterozygot, normal.

Following Rüdin further in the discussion of this question on inheritance are given diagrammatic in Figs. V and VI, illustrating respectively the inheritance proportions, where abnormalities are dominant and where abnormalities are recessive. These diagrams give a better explanation of the proportions that are usually expressed by the formulas *DD, DR* and *RR,* which are given above.

Rüdin states that individuals with identical types of ancestors, which to all appearance, have identical characters, notwithstanding this, can have the combinations of gameten that are entirely different. Thus, in Fig. VI, in the square marked 5 and 6, the children have identical types of ancestors, but the children are not the same, and seven of the eight children who externally appear to be the same have entirely different combinations of germ plasms. The opposite also holds true that individuals can have identical gameten combinations and at the same time have quite different types of ancestors. In Fig. VI, square 2 and 3, we find quite a difference in inheritance where abnormality is dominant or recessive. In families where the abnormality is dominant there will

be a great many abnormal progeny, as shown in Fig. V, with 17 abnormal individuals, while in Fig. VI there are only seven abnormal individuals in the progeny, and particularly in each and every generation and in each and every family where one parent

FIG. V. (After Rüdin.)
Inheritance Proportions in Dominant Abnormal.



● Abnormal, dominant homozygot. ◐ Abnormal, dominant heterozygot.
○ Normal, recessive, homozygot.

is abnormal; on the other hand, in families with the recessive Mendelian abnormality in much fewer individuals, and not in each and every generation and each and every family. This latter fact is of extreme importance, for an abnormality can skip two or even three or more generations.

Contrary to the rule in the dominant type of inheritance of an abnormality, we see in families, where abnormalities are of the recessive type, that external normal individuals are not always at the same time produced by normal germ plasm, for the external appearance of normality may cause errors to be made, and this rule is important when the question of marriage of relatives, cousins, etc. While they may appear absolutely normal, at the same time the tendency to abnormal characteristics may be present in the germ plasm of each individual, consequently, the children of such mating are much more liable to be defective through the inheritance of these latent abnormalities in the parents. Each and every normal individual from a family with a dominant abnormality is also of normal germ plasm. In normal individuals, from a family of recessive abnormalities, the same can also occur, but it is not absolutely necessary Fig. VI, square 3). Through different associations of matings and pairing the resulting proportions through the experiments in animal and plant life, the homozygous and heterozygous elements have been produced and accurately settled. Each and every abnormal individual of a family with a recessive abnormality is consequently of abnormal germ plasm. On the other hand, abnormal individuals have a dominant abnormality when one of the parents is normal, possesses also an anlage to normal. In cases of dominant abnormalities there is the danger only for the progeny of the abnormals, but for the normal progeny there is no further danger.

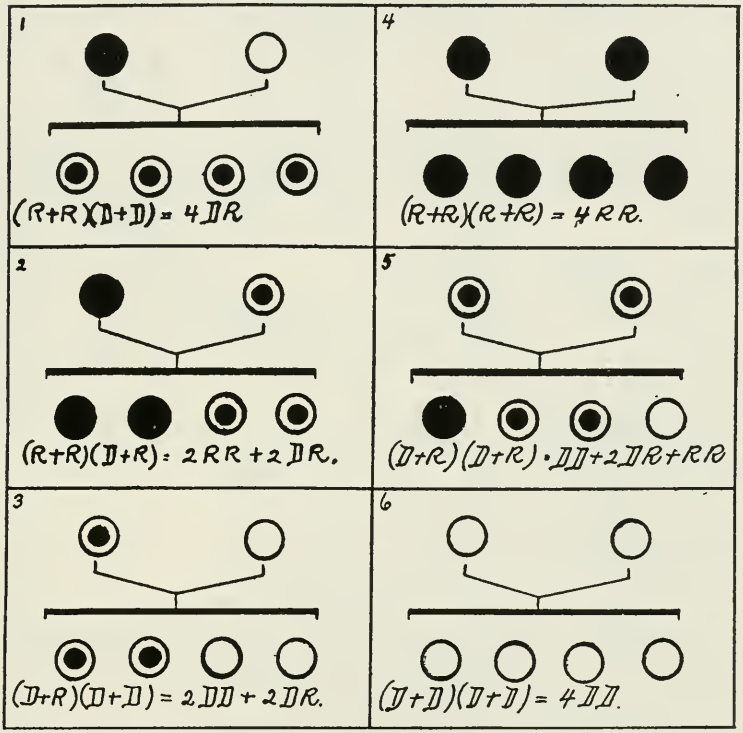
Rüdin gives us two main points from the standpoint of prophylaxis. 1. For families with inheritance of dominant abnormality, matings should be made only by normal members of a new stock, or with members of the same family. 2. In families with recessive abnormalities, matings can be made by all the individuals, but for the best results, certainly only the normal individuals, not only with new stock, but with the members of a normal new stock. These points that we have just spoken of can be said to be closely allied to the " mutation theory " of H. De Vries.

Rüdin discusses at length the Galton theory of inheritance of characteristics from ancestors, which views are utilized by Pearson, Darbishire and others, known as the Biometric School. Galton's law, concerning inheritance, is that a given person inherits one-half from the father and mother, from the grandparents one-

quarter, etc. That is, the given person is related one-half to the father and mother, and one-quarter to the grandparents, and one-sixteenth to the great grandparents. We have the following formula of the proportions of inheritance from the ancestors:

$$\tfrac{1}{2}+\tfrac{1}{4}+\tfrac{1}{8}+1/16+\ldots\ldots=1.$$

FIG. VI. (After Rüdin.)
Inheritance Proportion in Recessive Abnormal.



● Abnormal, recessive homozygot. ◉ Normal, heterozygot.
○ Normal, dominant homozygot.

Rüdin opposes this law of Galton, which has had such an effect upon the English school. He maintains that a given person does not inherit half from the father and mother, or one-quarter from the grandparents, but inherits distinct characteristics of the fathers or mothers or grandparents. In other words, from the Mendelian point of view, a person may inherit characteristics which make

him directly related to any one of his ancestors, or there may be no points of resemblance. Rüdin further discusses the complication which arises when one and the same abnormality is dominant for male members of a family, on the other hand, recessive for female members of the same family.

In Fig. VII is shown the well-known chart of Bateson, exhibiting the inheritance of color blindness. This peculiar form of inheritance occurs in other diseases, especially in hæmophilia. This form of inheritance was looked on as an exception to the Mendelian rule. By such charts analyzed closely, it is seen that they follow a fixed rule.

Rüdin gives, further, a large number of characteristics and traits in both the botanical and zoological fields. But of interest to us here are the characteristics often dominant in human individuals.

The Hapsburg lower lip in the male sex is almost exclusively dominant over the normal lips.

Single births dominant over twins and triplets.

Huntingdon's chorea dominant over normal.

Familial periodic paralysis over normal.

Porokeratosis over normal.

Night blindness over normal.

Progressive muscular atrophy over normal.

Hæmeturia over normal.

Ptosis familias over normal.

Normal dominant over amaurotic idiocy.

Many familial muscular diseases over normal.

Familial psoriasis and cerebella hereditary ataxia over normal.

Many forms of ichthosis palmaris over normal.

Normal dominates over albuminurea.

Brachydactyle or hyperphlange over normal.

A great many skin diseases dominant over normal.

Diabetes incipidis ever normal.

Some forms of glaucoma over normal.

Normal over retinis pigmentosa.

Color blindness in males over normal.

Hæmophilia in males over normal.

Pseudo-hypertrophic muscular paralysis (Gower's) over normal.

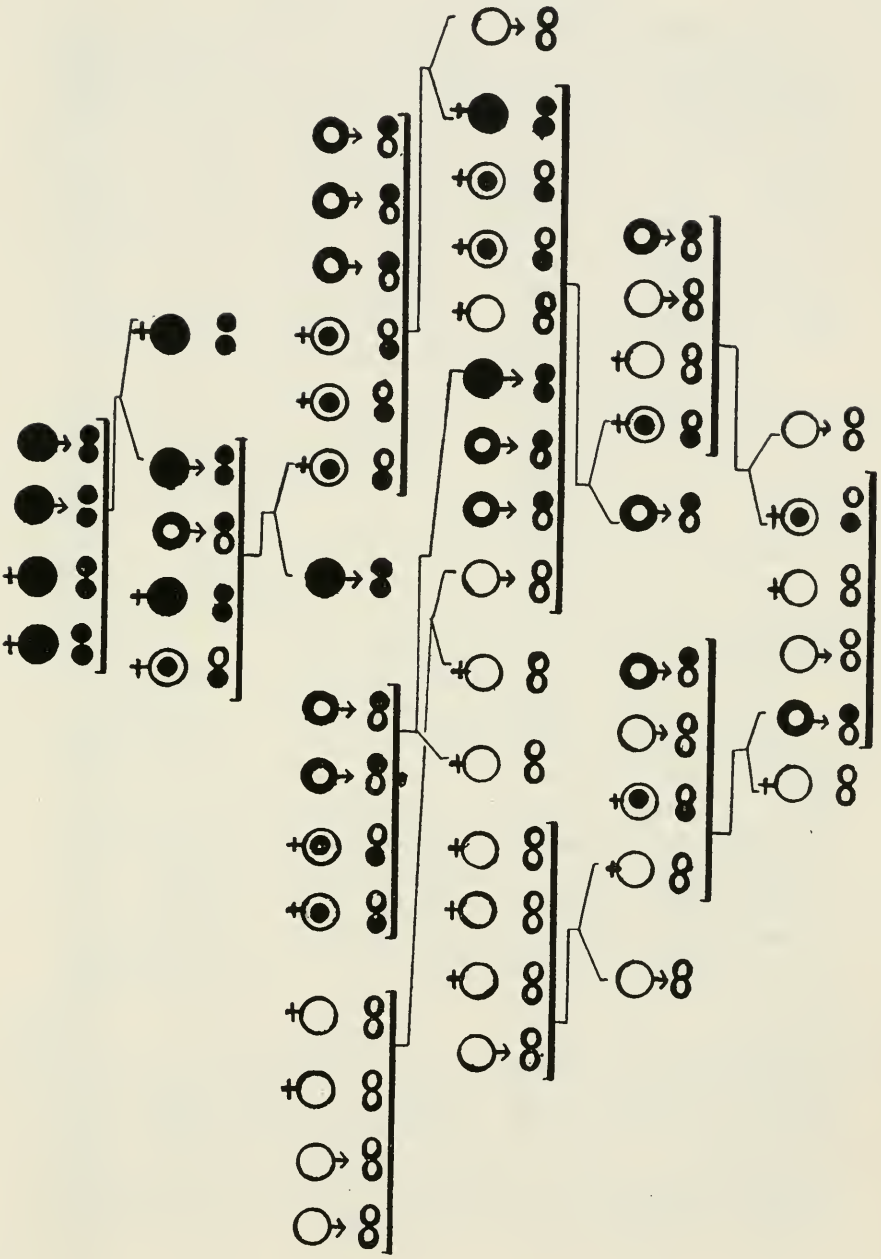


FIG. VII. (After Bateson.)
The Inheritance of Color Blindness.

The Problems in Psychiatry.

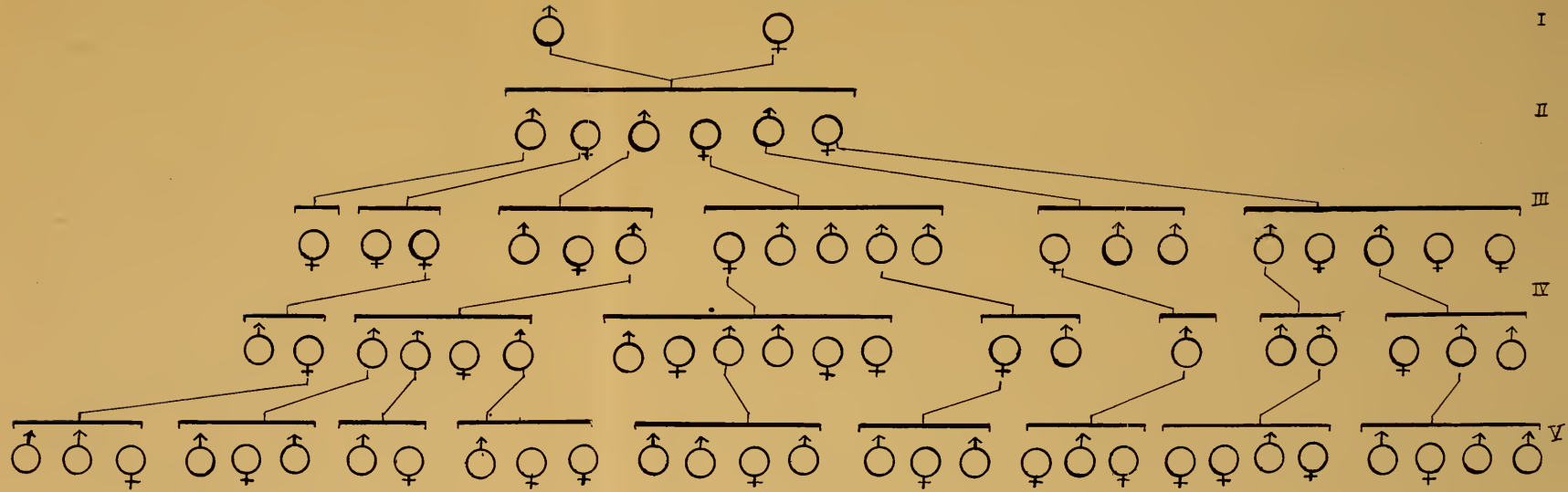
The problems relating to psychiatry will, therefore, be much more complex from our standpoint, for in a great many types external factors play a much more important rôle in the production of psychoses than the hereditary features. Especially is this true of the types which are due to the direct effect of toxins and poisons. Such is the case, for instance, in general paralysis, where we know to a certainty that the disease is not dependent upon heredity, but upon the effects of previous infection of syphilis. Delirious conditions, exhaustion psychoses, and, to some extent, manic-depressive insanity, might be produced by external factors which largely outweigh any effect of defective inheritance.

Among the problems met with by psychiatrists will be: 1st, study of direct inheritance of certain types. 2d, the effects upon the succeeding generations of the neuropathic constitutions as expressed by eccentricities, peculiarities, alcoholism, etc., in the parents. 3d, to what extent these factors are responsible for the occurrence of various types of psychoses in the progeny. 4th, the effect of the occurrence of certain types of psychoses in the ancestors upon the production of either similar or dissimilar types in the progeny.

Part of the work of the State Hospital, at Trenton, has been to collect as much accurate data as possible of the families and ancestors of patients coming under our care. It is a tremendous task to analyze this data, and often it is impossible to come to conclusions regarding the types of psychoses occurring in ancestors where we have only the description handed down from generation to generation. One has to be extremely careful not to be biased and make the cases fit certain laws. None the less, the value of such work is apparent when we consider the marked difference in the number of individuals about whom we now obtain information to the work previously done without the assistance of field workers.

While field workers are not trained psychiatrists, at the same time, we endeavor to have them attend the staff meetings whenever they are not in the field, to get some general idea of symptoms and diagnoses. In this way they become familiar with some of the important symptoms to be looked for, the age of the onset and whether individuals recover from their disease or become chronic,

FIG. VIII. (After Rüdin.)
Inheritance Chart.



and other points of importance. It is well that they do not know too much about diagnoses, as they might easily fall into the error of making certain diseases fit certain diagnoses, whereas, now their principal part is to collect *facts,* and then these facts are worked over and analyzed. When insufficient data is present no diagnosis is made and the case is considered as unclassified. We have been able to secure valuable data in a large number of cases, and will continue to make systematic investigations in all psychoses before giving any definite conclusions.

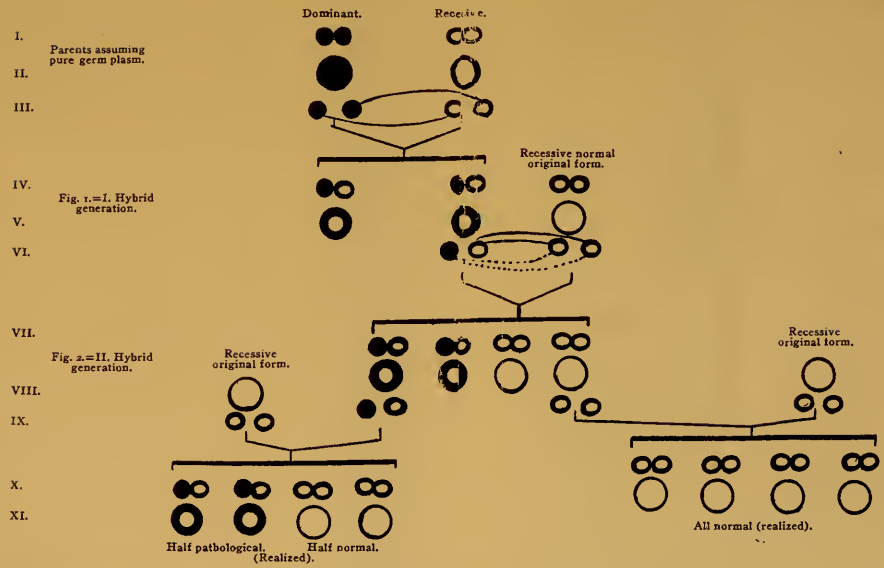
Rüdin states further that in the realm of psychiatry not enough work has been done to say with certainty whether these diseases follow the Mendelian laws or not, and he thinks it will be quite a long time before this question can be fully settled. He disagrees with Herron, that mental diseases do not follow any Mendelian laws, that they are neither dominant or recessive. He is inclined to think that the inheritance of dementia præcox follows recessive type, but the question cannot be settled at the present time. He is also inclined to think that manic-depressive insanity is a dominant type of inheritance. Although he thinks he has evidence which would substantiate these views, at the same time he is not willing to say that these diseases represent two separate types of inheritance. Many psychopathic states and defective conditions, he thinks, follow the same rule as in manic-depressive insanity. From the material at hand, he is inclined to think that intermediate types of inheritance, where there is a mixture in the offspring of the two distinct psychopathic conditions in the parents, is very seldom found. On the other hand, we see a certain similarity between psychopathic diseases in both parents and their offspring.

Rüdin enters into a lengthy discussion of the question of correlation, but for the present we will not get into a discussion of this principle.

He goes into a detailed discussion of the ways and means of systematic investigation of family histories of patients, and in Fig. VIII is reproduced his heredity chart. As will be seen, this method of charting differs somewhat from the method shown in Plate I. It is a question whether this is a simpler method of charting heredity than the former one. Instead of representing the matings by a line between two parents, Rüdin's method, as can be seen by his chart, is, to my mind, a better one, principally

FIG. X.

THEORETICAL EXPLANATION OF THE REDUCTION PHALANGES.



The following combinations are not realized in the reality, because the supposition of the pure germ plasm of the abnormal and the mated abnormals fails too in the actuality.

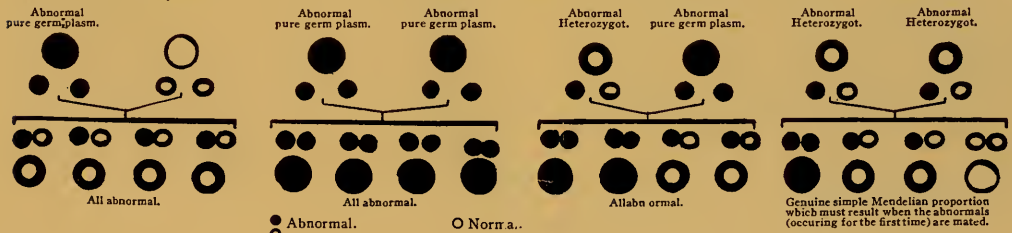


PLATE I.

KEY TO HEREDITY CHART, TAKEN FROM EUGENICS RECORD OFFICE, BULLETIN No. 2.

	Male.	Female.	
	□	○	No Data.
Red	E	E	Epileptic.
Black	F	F	Feeble-minded.
Green	I	I	Insane.
Violet	C	C	Criminalistic.

Other letters used in or around the squares or circles are:

A Alcoholic.
B Blind.
D Deaf.
M Migraneous.
N Normal.
Ne. Neurotic.
P Paralytic.
Sx. Sexually immoral.
S Syphilitic.
T Tubercular.
W Wanderer or confirmed runaway.

FIGURES.

Above the line—Order in the line of birth.
Above the square or circle—Individual reference number.
Below the square or circle—Age at time of death or date of birth or death.
In squares or circles—Number of individuals of that sex.

SMALL LETTERS.

b—Born.
† (d) inf.—Died in infancy.
† or (d) Died or dead.
m—Married.

LINES.

Solid—Connects married individuals and fraternities.
Dotted—Not married or illegitimate.
For display charts: Green—Paternal side, Red—Maternal side } of individual under study.
Violet—Connects related charts or individuals on more than one chart.

SYMBOLS.

Shows patient at institution reporting.

Miscarriage or stillbirth.

Institutional care (place under symbol).

because it allows the brothers and sisters of the family to be placed directly in series, while, on the other chart, it is found necessary to separate these brothers and sisters in order to get in all of the matings. Even when matings are represented by drop-

PLATE II.

ABBREVIATIONS FOR CHARTS FROM EUGENICS RECORD OFFICE, BULL. No. 2

To be used with full face symbols.

a, alcoholic insanity.	p, paranoia.
d, dementia precox.	s, senile dementia.
g, general paralysis of the insane.	
m, manic depressive insanity.	t, traumatic insanity.

To be written on chart.

bd Bright's disease.	*la* locomotor ataxia.
ca cancer.	*md* manic depressive insanity.
cb childbirth.	*np* neuropathic condition.
ch chorea.	*obs* obesity.
cr cripple.	*pa* paranoia.
df deformed.	*pn* pneumonia.
dp dementia precox.	*sh* shiftlessness.
dt delirium tremens.	*sm* simple meningitis.
dy dropsy.	*sb* softening of the brain.
ec excentricity.	*sco* scoliosis.
en encephalitis.	*sd* senile dementia.
go goitre.	*su* suicide.
gp general paralysis of the insane	*va* varices, varicose veins.
hy hysteria.	*ve* vertigo.
id ill-defined organic disease.	*x* unknown.
kd kidney disease.	*?* implies doubt.

ping one parent below the line, as is done at the Epileptic Village and the Training School, at Vineland, there is more or less confusion. In Rüdin's method one can see at a glance the number of brothers and sisters, and the proportion of those affected to the normals. The biological symbols used by Rüdin are not as clear as the symbols now in use, and shown in Plate I.

We have adopted at this hospital a combination between these two methods, in which the only change is the method of mating individuals. All other symbols are the same as the original method shown in Plate I.

Rüdin further gives some very complete printed blanks to be filled out by members of the family of the patient, or by a field worker. These are very complicated, and go much into detail, and for our use are rather too bulky.

The work of Rüdin is especially valuable to those working in this field, as many valuable suggestions are given, methods outlined and the essential data to be obtained to make the work valuable. Many of the problems are discussed and methods of attack suggested.

The Work of the Trenton State Hospital.

It will not be out of place here to describe the methods used at this hospital, where we combine "field work" for the study of heredity with "after-care" work.

We have had for a year two trained field workers, supplied through the courtesy of Dr. Charles B. Davenport, to collect data in regard to hereditary factors in the family history of patients. We have not limited them to any certain line, but have insisted that all possible information in regard to relatives should be obtained. In one instance one field worker obtained information in regard to 3300 members of a family group. This family group was located in one of the northern counties of the state, and had intermarried to such an extent that only five distinct families were represented. Of this number, 76 were insane, 22 were patients in the State Hospital, at Trenton, 14 in other hospitals and 40 not committed. Following is a list of the various abnormal individuals:

Sexual offenders	50
Epileptics	5
Alcohol	46
Feebleminded	13
Cancer	19
Sarcoma	1
Blind	2
Congenital defective	1

In practically every case investigated it is possible to obtain some information in not less than 200 members of a family, and sometimes a great many more. The field workers have found no difficulty in obtaining this information, and, without exception, they have received courteous treatment from the individuals whom they have visited. We find that the families are much interested in the work and will give all the information possible. The field worker becomes acquainted with the patient, and she talks with the patient before going to the family, and carries messages back and forth, and in this way establishes friendly relations. They spend on an average of fifteen days a month in the field. The rest of the time is spent at the hospital, writing up histories and making out charts. They do not attempt to make diagnoses, but take down all that is given by the relatives. Whenever the family physicians know anything about the family, they are visited, and their opinions also noted. Where relatives have been in the hospital, reference is made to this and a diagnosis made from the records when possible. When relatives have been in other hospitals, either in this state or in any other state, we have endeavored to obtain a copy of the records of these institutions.

The patients in the hospital are catalogued according to communities, towns, cities, etc., and when the field worker goes into a certain district she has the names of the discharged patients who are living in that community. A visit is made to these discharged patients to learn something as to their condition, and often the environment is such that it is necessary to report this to the hospital, and then advice can be given to the family as to the right method to pursue to prevent the recurrence of an attack.

This "after-care" work is a very important part of our field work, and has resulted in much good to discharged patients. Several times during the year the field workers devoted all their time to looking up discharged patients. Besides looking up the heredity in families, they inquire into the habits, domestic relations, occupation, and any other factors which are wanted by the physicians. In certain cases, where the statements of the family were questioned, the field workers had to go personally into the community, and they were able to prove or disprove these statements.

We have now collected a large number of pedigrees, averaging 200 or more to a family. It is not my purpose to go into any close

analysis of these charts, but a few are given merely to show the progress of the work.

In Chart I we have a pedigree of a case of neurasthenia. The generations are given on the left-hand side of the chart. Each individual is numbered according to that generation. A transcript of the notes is given in this case to show the method of the work.

To summarize, we have a patient, a neurasthenic, one of three children, a sister of whom was epileptic and a brother nervous. The father was a manic-depressive case, who committed suicide, and the mother was neurotic. Father's family is apparently of good stock. The mother's family, however, shows marked defects. Maternal grandfather was a neurasthenic. Maternal grandmother was also a neurasthenic. One maternal uncle epileptic, and another alcoholic. Maternal aunt suffered from manic-depressive insanity, but recovered. An epileptic aunt has one epileptic boy. One of patient's great-great grandmothers, on the mother's side, was insane for 30 years, died at the age of 60, following the death of child, from which she did not recover. In the maternal grandparents' line there is a good deal of nervousness and neurasthenia. IV-39 was a patient in this hospital, manic-depressive insanity, recovered, and married a former patient. His wife had another attack, and recently the man committed suicide. His father was also a manic-depressive case, and committed suicide.

Following are the notes made by the field worker in this case:

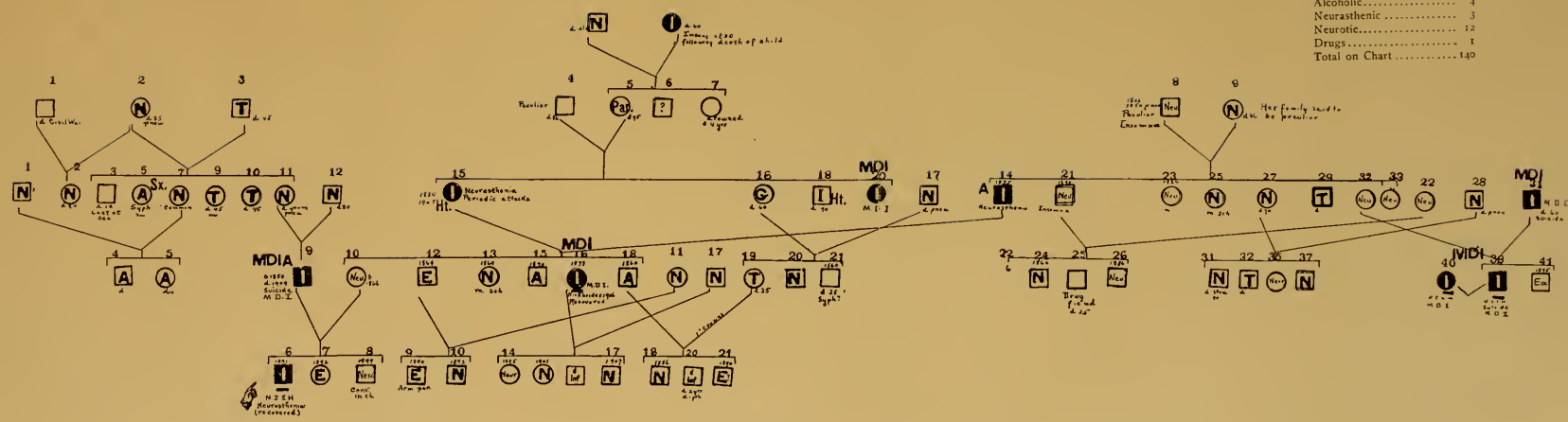
H. H., neurasthenia (sexual) on constitutional basis. Admitted July 24, 1911. Age 20.

H. H., born in 1891, oldest child of John G. H. and Harriet S. H. He has always been extremely nervous since early childhood, had never been like other children and has always been a source of constant worry to his mother. He has always read "deep" books and stayed indoors to read them. Many of these books were quack medical books. He is the oldest of three children. The next child, a girl, Helen, is nervous and has suffered from convulsions after eating something which did not agree with her. It is perhaps epilepsy, as a brother of her mother suffers from epilepsy. The youngest child, a boy, is very nervous. There is a strong neuropathic tendency throughout the family, past as well as present generations. There is no insanity in the father's family, though there is a tendency toward sex perversion on the paternal grandmother's side. The patient's father himself committed suicide following two years drinking heavily after business reverses. The mother's family is all neuropathic, very few normal individuals to be found in the entire history. A great many of the people not committed were in a much more dangerous condition than the patient him-

HH

CHART L

Insane	9
Epileptic	4
Alcoholic	4
Neurasthenic	3
Neurotic	12
Drugs	1
Total on Chart	140



self. The maternal grandparents and great grandparents were eccentric, a great-great grandmother was insane, a maternal aunt was insane and recovered, a maternal uncle epileptic, a maternal great aunt and uncle insane, though never committed. A second cousin insane and, in this institution, recovered. Many others neurotic.

I. Nothing known of this generation of H.

II-2. E. P., married first a man by the name of H. He was killed in the Civil War. She died at 85 of pneumonia, and was normal always. She had one daughter by this first marriage, Eliza.

III-2. E. H., married C. F. (III-I), of W., N. J. She died at the age of 94 years. They had no children, but brought up J. H., the patient's father.

II-3. J. B., E. P.'s second husband died at 45, T. B. He was normal mentally. They had six children, James, Lydia, Angeline, Harris, Jane, Martha.

III-3. James, lost at sea, age 19.

III-5. Lydia B, who is still living. She is a *sex offender,* is alcoholic, has had syphilis and is described as a hard, bad woman. She married E. F. (II-4), a politician, of N., who is dead. Cause unknown. They had one child, Lizzie.

IV-2. Lizzie, who of T. B. She married and had one child, that died at birth.

III-7. A. B., still living, married and had two children. She is described as being "common." Her children were Edward and Harriet.

IV-4. Edward, very alcoholic, died of asthma. Married.

IV-5. Harriet, very alcoholic, died of dropsy while still young. Married. No children.

III-9. H. B., died at 45 of T. B. She married E. J., of N. They had one son.

IV-7. M. J., born 1865, married, and had four children, Raymond (LV-2-5), Perry, Anna May, Mildred, all normal children.

III-10. Jane, died at 45 of T. B.

III-11. M. B., died very young of pneumonia. She married.

III-12. E. H., of N. He died of old age at 80. He married again and had four children. Nothing known of them. They had one son, J. H., the patient's father.

IV-9. J. H., born 1850. He was a newspaper man, and, following business reverses, took to drinking heavily for two years. He quit drinking, but felt the disgrace so keenly he *committed suicide.* He must have been temporarily insane, for he had made all his plans for the next day—died 1899—suicide. He married Harriet S.

IV-15. Charles, born 1870, works on railroad, is normal, but drinks quite heavily.

IV-16. Louise S., born 1873. She was always nervous, even as a child. She was finally committed to *Hospital*, 1896. She remained there three or four months, but it was a year before she fully recovered. *Diagnosis, melancholia.* She married S. C., a school principal of W. They have had four children, Mildred, Frances, Cecil, and a baby that died at 11 days.

V-14. Mildred, born 1895, very *nervous.*

V-15. Frances, born 1901.

V-16. Boy, died at 11 days.

V-17. Cecil, born 1907, normal.

IV-18. David S., born 1860. Periodically alcoholic. He married Ella K., a first cousin, who died of T. B. at 35 years. They had three children, Clarence, Tuttle, Lester.

V-18. Clarence, born 1886, a government life saver. He is delicate, but normal.

V-19. He married G. T. They have one daughter.

VI. Ruth, born 1909.

V-20. Tuttle, died of diphtheria at 2 years.

V-21. Lester, born 1890, a farmer at F. He had *severe convulsions* up to the time he was 14 years. None since.

I-1. S. L., died very old. Married H. T.

I-2. H. T., *insane.* She went insane following the death by drowning of her 4-year-old daughter. She was kept at home and was mildly demented. Died at 60. They had three children, Margaret, Charles and the girl who was drowned.

II-5. Margaret L., died at 75 of paralysis. She married (II-4) F. C., a miller of F. He was of rather weak character and *was peculiar.* Died at 85 of old age. They had four children, Hannah, Mary, John, Anna.

III-15. Hannah (patient's grandmother), married C. S.

III-16. Mary, died at 60 of *cancer* of stomach. Married W. K. (III-17), who died of pneumonia. They had three children, Ella, Frank, Edward.

IV-9. E. K., married her first cousin, D. S. (IV-18). She died at 35 of T. B.

IV-20. Frank, living, normal.

IV-21. Edward, died at 25 in 1885, after 3 or 4 years sickness. He lost use of limbs, flesh rotted away, thinks it may have been syphilis.

III-18. John, died at 70 of heart failure. *He told lies, did not seem to know they were lies.* Married H. B., who is still living. No children.

III-20. Anna C. *She is subject to depressed spells. Has always posed as a martyr,* knowing she will be rewarded hereafter. Tells malicious lies about the neighbors and friends, seemingly believing them to be true.

II-8. D. S., born in 1808, died in 1850 of pneumonia. He was always nervous and peculiar. Suffered from *insomnia* always. He married (II-9) H. H., who died of old age at 86. She was normal, though her family is said to have been queer. They had fourteen children, Charles, Kate, Elizabeth, Jacob, Anna, Harriet, William, Helen, and six that died in infancy.

III-14. Charles (patient's grandfather).

III-21. Jacob, born 1832, still living. He is a newspaper man, an editor at times. He has been *neurotic* for years. Has suffered from *insomnia* and is *childish now*. He married Martha J. M., who is very nervous. Still living. They had nine children, Robert, Albert, Warren, and six that died in infancy.

IV. H. S., born in H. in 1866, a daughter of C., and H. S. She is of a highly *nervous temperament*. Worries over H., over the other children, etc. She recognizes plainly the neurotic tendency in the family. They have three children, Hamilton, Helen and Jack.

V-6. H. H., born 1891. *Admitted to N. J. S. H.* in July 1911, diagnosed *neurasthenia* (sexual) *on constitutional basis*.

V-7. H. H., born 1892. Nervous temperament. She has had severe convulsions since infancy whenever anything she eats disagrees with her. Had one severe convulsion, summer, 1911. *Epilepsy*.

V-8. J. H., born 1897. Is extremely *nervous*. Had convulsions while teething.

III-14. C. S., born 1835. Lived in H., a mason by trade. He has always been ugly and *irritable, "devilish"* at times. Seemed to try to torment his family, drove his wife nearly insane, with his tormenting. Has been a steady drinker all his adult life. He married (III-15).

III-15. H. C., of F. She was one of four children. She was born in 1834, died in 1905 of dropsy and heart trouble. She was very nervous, almost, if not, insane, at times, when she became so excited that she had to be restrained. She and C. S. had six children, Harriet, Walter, Jennie, Charles, Louise, David.

IV-11. Walter, born in 1864. He is an electrician in N. Has suffered from *epilepsy* all his life. Is rather irritable and hard to get along with. He married L. H., who is strong and well. They have two sons, George and Ben.

V-9. George, born 1890, had severe convulsions as a child, one arm has been paralyzed since he was 6 or 7 years of age. *Epilepsy (?)*.

V-10. Bert, born 1892, normal.

IV-13. Jennie S., born 1889. She is normal in every way. Married Richard N., of F., N. J., painter and decorator. Three children, Joseph, Ethel, Richard.

V-11. Joseph, born 1894.

V-12. Ethel, born 1897.

V-13. Richard, born 1902.

IV-24. Robert, born 1860. A musician in P. He married J. V., of L. No children.

IV-25. Albert, died at 25. He was a dentist, and became a morphine fiend, which drug probably caused his death.

IV-26. Warren, born 1886, a farmer. Very nervous. Married. No children.

III-23. Elizabeth, born 1826. *Very nervous.* Married E. T., who died of Bright's disease. No children.

III-25, Kate, still living. Married R. O., of H., N. J. They had several children, only two lived to grow up, Harry and Richard.

IV-31. Harry, a traveling man, married M. S., who died of stomach trouble. They had two children, a girl and a boy.

IV-33. R. O., died when a young man, of Bright's disease. He was a doctor.

III-27. A. S., normal. Died at 70 years of old age. She married T. T., who died of pneumonia. They had four children, Melville, Kate, Frank, Edward.

IV-31. Melville T, died of stomach trouble. He married M. C. They had two children, Melville and Anna.

IV-33. Kate, very nervous, married C. F., who died of appendicitis. They had two children, Norman and Charles.

IV-37. Frank, normal, married Olive —. They have 5 children.

III-29. William, died of T. B., while still a young man.

III-32. Harriet S., died of uræmic poisoning. She was always *neurotic,* always doctoring for her nerves. If she had not been so well taken care of would probably have been a neurasthenic case. She married W. S., a school principal, who *committed suicide* at 60 years. They had two children, Forrest and Lyle.

IV-39. F. S., admitted to N. J. S. H. in 1907. Discharged in 1908. A case of manic-depressive insanity, manic attack. Died March, 1912, suicide. He married E. D. C., whom he met while in the hospital. She was admitted in 1906, discharged in 1908, manic-depressive insanity, depressed type. Readmitted Febuary, 1912, M. D. I., manic type.

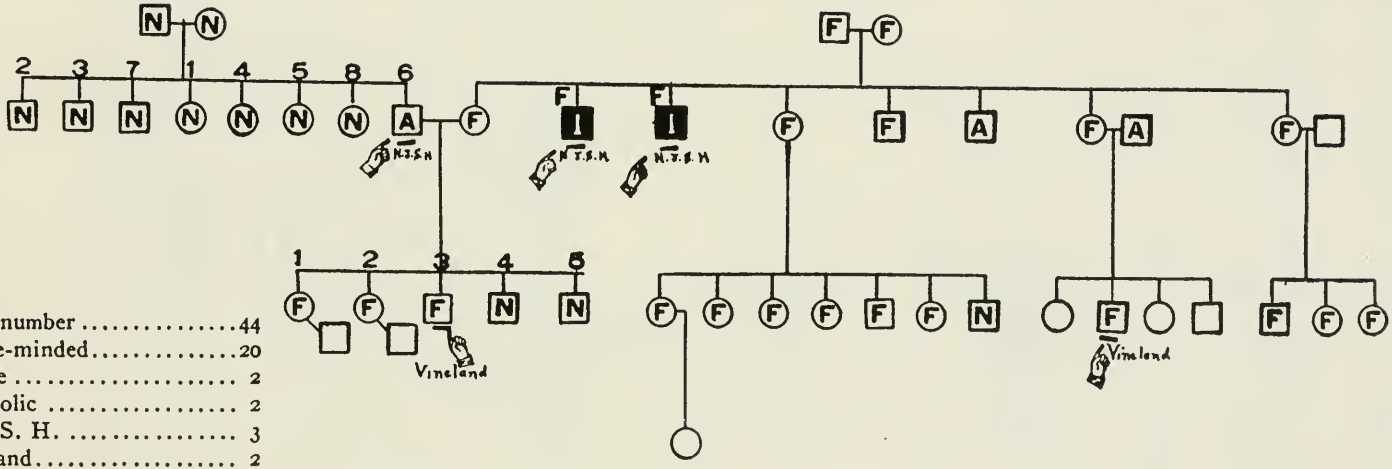
IV-41. L. S., born in 1875. He is a civil engineer, and is rather *eccentric.*

III-33. H. S., born in 1850. She is *very nervous.* Unmarried.

Chart II is an illustration of the method charting that is used by the Eugenics Record Office.

CHART II.

BB

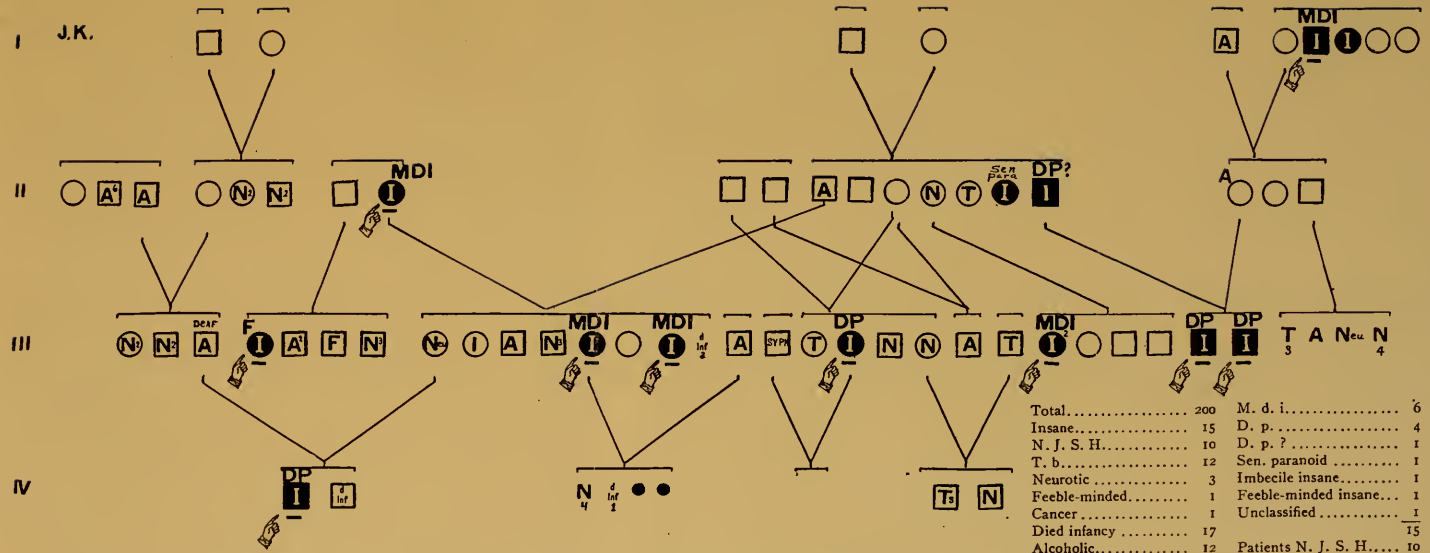


Total number	44
Feeble-minded	20
Insane	2
Alcoholic	2
N. J. S. H.	3
Vineland	2

There are 42 individuals in this chart, whereas, Chart I has over 70. This shows the inheritance through marriage of two feebleminded individuals. Nine children were born to this family, all of which are feebleminded, one alcoholic. Two of these children are inmates of this hospital at present. One girl of this feebleminded pair married a man, who was an alcoholic, but his family was normal. As the result of this union three are feebleminded and two normal. One feebleminded child is in Vineland. Another girl married a man, who was an alcoholic, and has one feebleminded child at Vineland. Four members of this group are now cared for by state institutions. There are altogether 22 feebleminded progeny from the original mating.

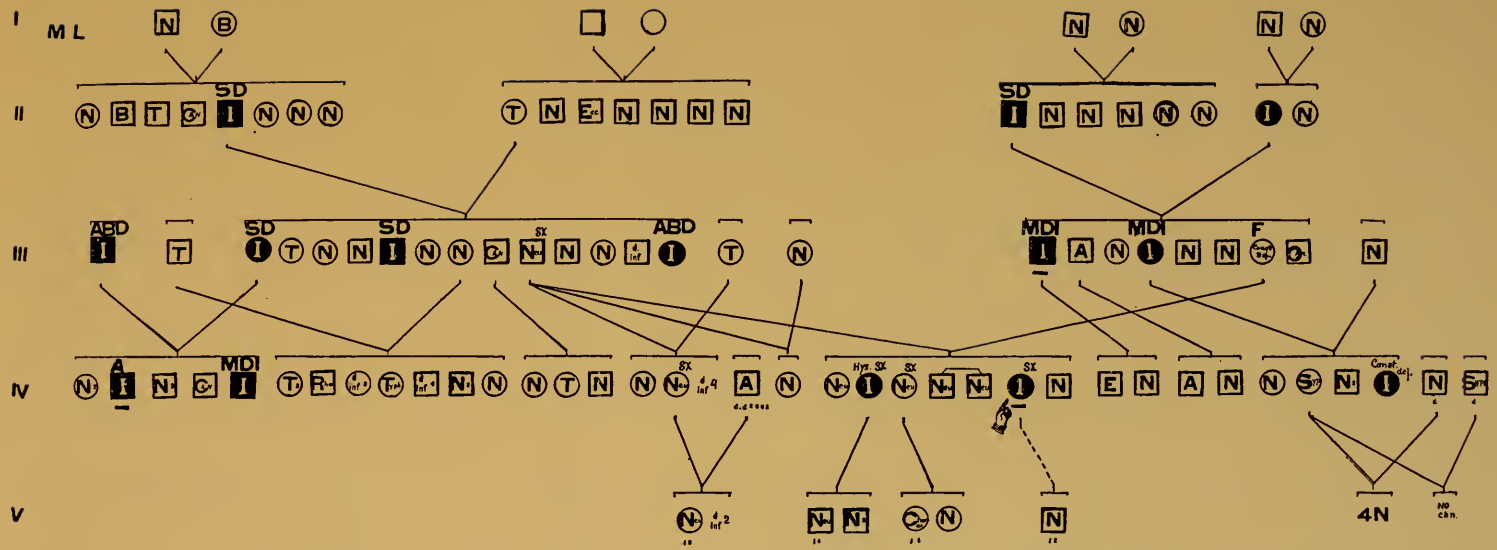
Chart III is a summary which represents 200 individuals, 15 of which were insane. Ten were in the Trenton State Hospital, and 12 were tubercular, three neurotic, one feebleminded. Seventeen died in infancy. Nineteen were alcoholic. Of the psychoses, we have six manic depressives, four dementia præcox, one questionable dementia præcox, one senile paranoid condition, one imbecility, one feebleminded and one unclassified. The paternal line is fairly good, with the exception of alcoholism. The maternal line, on the other hand, is very much affected. The mother is neurotic. One sister was a border-line case. There are two sisters manic-depressive. The mother had nine living children. Three died in infancy, making a family of 12. The mother was insane, had manic-depressive insanity, from which she recovered, and is now living at the age of 83. The father was alcoholic, had a sister who was a senile paranoid condition, and a brother dementia præcox. This brother had two children, both of which are dementia præcox, and inmates of this hospital. He married a woman put down as peculiar. A maternal cousin is a case of dementia præcox in this hospital. In this family, out of five individuals who were insane in the grandparents or great grandparents, only two were in institutions, while all the cases that were insane in the parents' children were committed to institutions. This fact will be found to run through all our charts, and one can conclude that in the preceding generations the percentage of cases who were insane and who were committed to an institution was much smaller than the percentage of the same class in present generation.

CHART III.



Total	200	M. d. i.	6
Insane	15	D. p.	4
N. J. S. H.	10	D. p. ?	1
T. b.	12	Sen. paranoid	1
Neurotic	3	Imbecile insane	1
Feeble-minded	1	Feeble-minded insane	1
Cancer	1	Unclassified	1
Died infancy	17		15
Alcoholic	12	Patients N. J. S. H.	10

Patient is d. p. Father alcoholic, mother neurotic. Paternal line alcoholic. Maternal line: Grandmother m. d. i.; grandfather alcoholic; 2 m. aunts m. d. i.; 3 m. 2nd cousins d. p., 1 m. d. i. and 1 f. and insane; 1 grand uncle d. p.; 1 grand aunt senile paranoid.



Total.....371	Insane 14	Syph..... 2	Deaf 2	Alc. ins..... 1	Con. def..... 1
Abnormal..... 73	Alcoholic 6	Blind 2	Cripple..... 2	Sen. dem..... 2	Ar. scl..... 3
Normal 298	T. b.....16	Cancer 5	Con. def..... 2	Sen. psyc..... 3	M. d. i..... 2
D. Infancy.... 16	Sx. 11	Epil..... 1	Neur.....10	Traum. psyc.... 1	Psychas. 1
	47	10	16	7	7

Patient is psychasthenic.
Father is neurotic and sx., mother constitutional defective.
Paternal line: 3 senile dementia, 2 arteriosclerotic, 1 m. d. i., 1 alc. ins.
Maternal line: Grandfather senile dementia, grandmother insane, 2 m. d. i., 1 f. insane, 1 epileptic.
Fraternal line: 7 neurotic, 4 sx., 2 insane.

This has an important bearing on the apparent increase in the number of insane in institutions at present, for I think I can be definitely shown that a large proportion of those who were insane in the community in previous generations were kept at home.

Chart IV represents a family of 371 members, in which 73 were abnormal in the following proportions: Insane, 14. Alcoholic, six. Sexual offenders, 11. Syphilitic, two. Blind, two. Cancer, five. Epileptic, one. Deaf mutes, two. Congenital, two. Constitutional defects, two. Neurotics, 10.

Diagnoses of the insane are as follows:

Alcoholic insanity	1
Senile dementia	2
Senile Psychoses	3
Senile trauma	1
Senile defective	1
Arteriosclerotic brain disease	3
Manic-depressive insanity	2
Psychasthenia	1

The patient represented by three asterisks was psychasthenic. She has six brothers and sisters. Two brothers, twins, and neurotic. Two sisters are neurotic, and one sister insane, with diagnosis of hysteria. We find that the mother was a constitutional defective. There were two brothers and sisters, each manic-depressive insanity, and the maternal grandparents were both senile psychoses. The father was neurotic or psychopathic sexual individual. He was married three times. He had two sisters. One was senile and the other arteriosclerotic. One brother suffered from senile psychosis, due to head trauma, at the age of 50. There were 13 brothers and sisters in this family. Three could be classed with the senile psychoses, and we find that the father of this family died at the age of 60, of senile dementia, while the mother came apparently from normal stock.

This chart illustrates a very important point, that is, the hereditary features of senile psychoses. Here the diagnosis is not made merely on old age, because there are in this family thirteen normal individuals living at the ages of 70, 93, 60 and 85. This tendency to senility in this family seems to be in the proportion of three to twelve, 13 children dying in infancy.

Another significant fact is the tendency in succeeding generations to develop manic-depressive insanity and psychopathic states.

Chart V illustrates inheritance in a case of dementia præcox. The patient was one of six children, three of which died in infancy, one was neurotic and one normal. The father was a case of manic-depressive insanity, recovered, living at the age of 62. He is one of six children. One brother has arteriosclerotic brain disease, three normal, one sister neurotic. Patient's maternal grandparents, the grandmother's line, is apparently normal. His paternal grandfather was a constitutional defective, died at 66, of arteriosclerosis. The grandfather has 12 brothers and sisters. One brother is put down as melancholy. One died at the age of 42, had sunstroke, and died insane. One was a constitutional defective, died at the age of 19. There were five affected individuals in this group of 13. Seven could be put down as normal, one neurotic. The mother of the patient was neurotic. She was one of fifteen children, seven of which died in infancy. Three were normal and one had harelip. The maternal grandmother was put down as insane. The maternal grandfather apparently normal line. We have a summing up, then, of a total of two hundred ninety-two. Insane, fifteen. Epileptics, four. Feebleminded, one. Neurotic, five. Harelip, one. Syphilis, two. Sexual offenders, two.

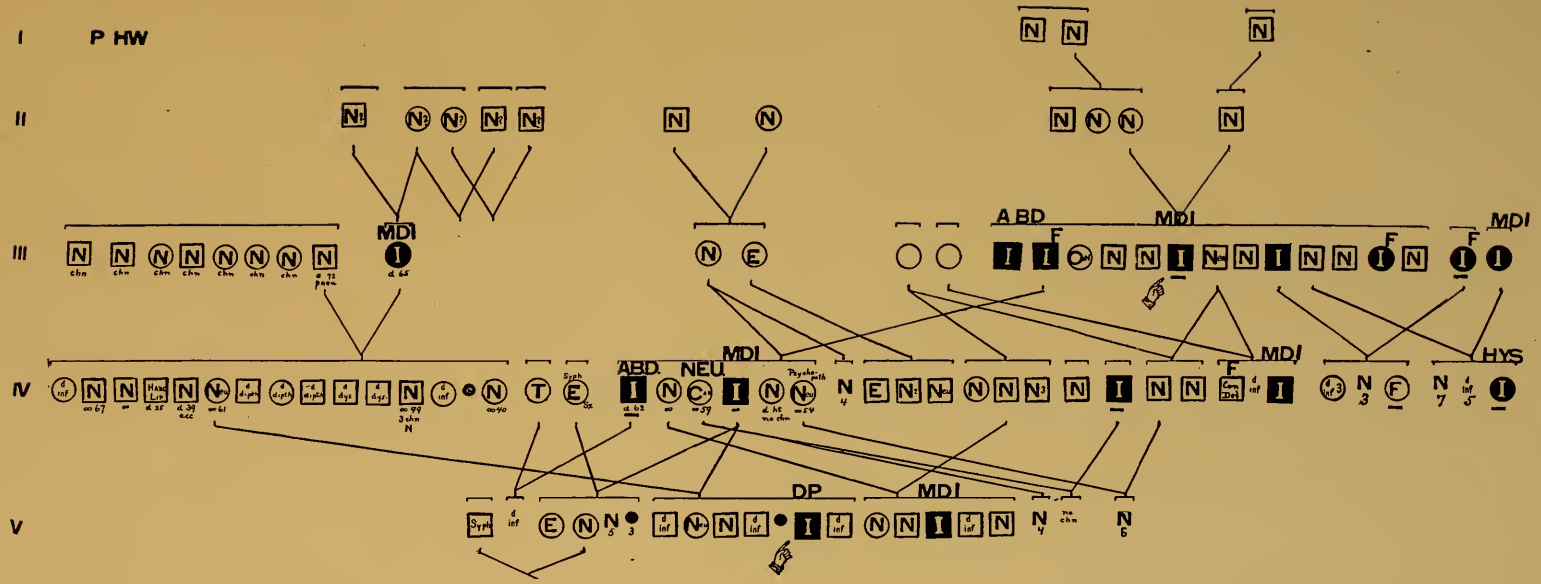
Diagnosis of those insane are as follows:

Constitutional defective	2
Melancholia	1
Dementia præcox	1
Psychoses following sunstroke	1
Hysteria	1
Manic-depressive insanity	3
Arteriosclerosis	2
Depression	1
Feebleminded	1
Unknown	2

Conclusions.

We have not attempted to analyze these charts carefully, but they are given merely to illustrate the progress of the work, and also to illustrate what a difficult task the analysis of these charts means. Frequently, when a point in question is necessary, the field worker visits the family again to clear up these disputed points.

CHART V.



Total.....292	Art. scler..... 2	Epilepsy 4	Patient d. p.
Const. def..... 2	M. d. i..... 3	Feeble-minded 1	Father m. d. i., mother neurotic, m. grandmother m. d. i., paternal grandfather insane (f.).
Melancholy 1	Depression..... 1	Neurotic 5	Maternal line: Clear, except 1 m. d. i., 1 neurotic.
D. p..... 1	Feeble-minded ins..... 1	Hare lip 1	Paternal line: 5 m. d. i., 2 arteriosclerotic, 3 feeble-minded insane, 2 feeble-minded, 5 neurotic, 3 epileptic, 1 hysterical.
Sun stroke 1	Unknown..... 2	Syphilis..... 2	
Hysteria 1	Insane.....15	Sx..... 2	

In this paper no attempt has been made to give any definite conclusions regarding the hereditary factors in the various psychoses. We have reviewed some of the most important work done so far, and outlined the methods to be pursued to obtain the best results in future work.

We have also spoken of some of the difficulties to be met with, especially when analyzing the material as it comes from the field workers. It is again well to emphasize the necessity of maintaining an open mind regarding these problems, and not to be too biased in attempting to make the facts fit the Mendelian laws. At the same time, we recognize that a comprehensive knowledge of the laws will assist us materially in analyzing our data and in arriving at practical conclusions. We also feel that much valuable material will be obtained which will aid us in solving the problems of prophylaxis and prevention of mental diseases. We hope that other hospitals and institutions will adopt this method of studying these important questions.

I wish to express my thanks to Miss Florence I. Orr and Miss Elizabeth P. Moore (field workers at this hospital) for their valuable assistance in making charts and furnishing valuable data for this paper.

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